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REVIEW OF RESEARCH ON FLASH BLINDNESS, CHORIORETINAL BURNS, COUNTERMEASURES, AND RELATED TOPICS

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irradiance and which may cause functional impairment of visual acuity, either permanent or transient. In the section of this report which reviews human exposures, a visual [illegible] even though no lesion can be seen during ophthalmologic examination. Between the levels which produce reversible dazzle and those producing ophthalmoscopically visible lesions, functional irreversible impairment of photoreceptors can take place. Chan, Berry, and Geeraets (27a) studied the alterations of soluble retinal proteins due to thermal injury using a micro agar electrophoresis technique. The amount of altered protein was greater for long exposures (500 ms) than for short exposures (175 μs), and can be detected 2 mm from a lesion produced by an exposure 40% above threshold. The extent of the thermal injury and its relationship to pulse duration is compatible with the concept of heat conduction as a significant modifying factor in the production of chorioretinal burns. McNeer, Ghosh, Geeraets, and Guerry (119a) noted electroretinographic (ERG) changes with large image exposures 50% below threshold for visible burns. The energy level which produced the ERG changes was computed to be well above that needed to alter soluble retinal proteins. Geeraets, Burkhart, and Guerry (74a) reported that histological preparations did not show alterations with exposures below threshold for ophthalmoscopically visible lesions. However, impairment of enzyme activity (DPN diaphorase and succinic dehydrogenase) could be detected at levels 10 to 15% below threshold for visible lesions.

C. Production, Histopathology, Clinical Manifestations, and Symptomatology of Chorioretinal Burns

There exist a large number of clinical reports on eclipse retinitis or solar chorioretinal burning. These reports are pertinent to chorioretinal burns produced by atomic fireballs and, although quantitative aspects of exposure time and energy absorption at the retina are lacking, these cases provide a large number of clinical reports regarding the symptomatology

and rate/degree of recovery of the human retina following the production of a thermal chorioretinal lesion.

1. Eclipse Retinitis (Sun Blindness, Solar Chorioretinitis)

The incidence of lesions caused by direct viewing of the sun is particularly great during an eclipse, and since the mechanisms involved may be the same for either viewing the sun or a nuclear detonation, one would expect similar histopathological and symptomalogical changes. The literature on solar retinitis is extensive, so the findings of a number of clinical reports have been summarized in the following paragraphs.

Patients report the rapid development of symptoms one to four hours after exposure, including sensations of "tearing", "smarting", "blurring", "clouding", and "dazzling". In more severe cases, loss of vision was pronounced but some improvement was noted in time. The symptoms described by some patients are similar to the subjective impairments of [illegible] blindness. The majority of the clinical reports were based on examinations which occurred months after exposure, with derangements of vision and loss of visual acuity ranging from 20/30 to 20/200. Lesions observed on the macula vary from small, single holes burned in various shapes to large holes. A number of cases were found with double large holes or multiple small holes. The lesions in the macula were easily visible and had the characteristic appearance of a deep crimson crater, irregular in shape with sharply defined edges and usually surrounded by a soft cloud of pigment. According to Agarwal and Malik (1), the severity of the lesion varied with the amount of energy absorbed and such lesions may be classified into four grades based on ophthalmoscopic changes:

(1) loss in acuity, macula apparently normal

(2) macula congested and surrounded by edematous area of retina

(3) a grayish-white patch surrounding the fovea which, in turn, was ringed by black pigment

(4) a macular hole and gross pigmentary change.

The 56 human subjects in this report experienced a variety of symptoms including one or more of the following: metamorphopsia, disturbances in color vision, translucid scotoma, photophobia, and persistent after-images(1). In the severe burns, absolute and positive scotoma developed with a gross diminution of vision. The basic pathological defect was considered to be similar to angiospastic retinopathy (i.e., vasospasm causing dimunition of retinal nutrition leading to localized ischaemia which, in turn, could bring about degenerative changes).

Treatment is briefly described in a number of the solar retinitis papers but, in general, the value of these clinical reports is to be found in the gross descriptions of the burns, symptomatology, and long-term alerations seen months or years later. Most subjects noted some visual recovery several days to several months later,, but permanent losses in visual acuity were always associated with macular burns. Some patients learned to compensate for this defect by training themselves to utilize the undamaged portions of the retina (1, 26, 36, 37, 44, 52, 60, 67, 101, 113, 160).

2. Photocoagulation by Optical Devices (Photocoagulators, Lasers)

Another source for examining effects of intense concentrated light on the human retina comes from studies utilizing a photocoagulator to treat human patients for detached retina and other superficial abnormalities of the retina. The earlier work in this area has been reviewed by Myer-Schwickerath (130).

Curtin and Norton (43) reported on the histological effects of supra-threshold treatement with the Zeiss photocoagulator. Early pathological changes seen in the area of photocoagulation revealed extensive coagulation necrosis in all retinal layers and choroid, with the sclera being unaffected. The most extensive damage was found in the pigmented epithelial layer, and choroidal vessels were engorged with intravascular thrombo-emboli. The endothelial cells of the choriocapillaries showed necrosis, as did the

walls of other larger blood vessels. Subretinal fluid shifts caused dissection of the sensory retina from the pigment epithelium peripheral to the site of burn (adjacent to focal spot). Disruption and scattering of the pigment was seen and necrotic changes were apparent in the rod and cone layer, the outer nuclear layer, and the outer plexiform layer. Choroidal vessels were dilated and engorged and a few inflammatory cells were scattered in the vascular layer. With more intense exposures, retinal rupture, hemorrhage, damage to the intimal limiting membrane, and leakage of subretinal fluid into the vitreous cavity were seen. By comparison with burns produced by direct viewing of the sun, the high energy photocoagulator lesions produced far more mechanical disruption of all retinal tissues.

Recently, Geeraets and Ham (74) reviewed the retinal effects of laser irradiation. They reported gross disruptions of the retina with both laser and photocoagulator high energy pulses to an area 0.76 mm in diameter on the retina. Retinal elements were forced out into the vitreous but no changes were seen in the subretinal tissues. Lesions were not uniform and hot spots in the same eye were seen, these hot spots being attributed to local differences in degree of pigmentation. Histologically, some areas developed more intense damage than others, but major effects were found in the pigment epithelial layer and in the receptor cells. Depending on the intensity of the lesion, the tissue responses varied. In moderate lesions, loosening of the retinal receptor cell layer from the pigment cell layer was common. This change was even seen in mild lesions, but with increasing intensity of heat input the lesion appears to have actually erupted. Subretinal hemorrhage and dispersion of pigment cells into the vitreous through tears in the retina were found. (In the area of the lesion, lipoidal globules were observed.*)

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*Pathological significance of appearance of lipoidal globules has not been established.

Zaret (200) has described experiments where, as laser energy input was increased, vaporization due to intense heating caused an explosion of the retina and marked dispersion of pigment granules. Three days post-irradiation, Zaret noted a central zone of destruction surrounded by a gray exudate and peripherally, a halo of pigment. Four to fourteen days later, hemorrhagic material began to disappear, and by thirty days scarring and some recovery were noted. Zweng and Flocks (202) using a modified hand-held photocoagulator reported almost instantaneous development of retinal hemorrhage following lasing. The lesion had a characteristic central red spot (bleeding) circumscribed by a halo which contained vapor bubbles up to 15 minutes post-exposure. Tears in the retina and dislocation of pigment were seen near the macular hole. Subsequently, scar formation took place, edema subsided, and circulation was restored, leaving a scar surrounded by a halo of atrophic retinal tissue.

Ham and his co-workers (84, 85, 86) have compiled probably the most abundant controlled data on chorioretinal burns in experimental animals. In addition to previously cited effects, they noted an almost immediate swelling of the nerve fiber layers and marked pyknosis of all nuclei in the inner and outer layer. The neurons of the ganglion cell layer were completely structureless and structure of rods and cones in the burnt area was lost. The pigment epithelium showed marked pyknosis, fragmentation, and chromatolysis. The choroid showed little change immediately post-burn. However, three days later, marked choroidal hyperemia was seen with relatively little leukocytic infiltration at that time. The subsequent histological alterations were similar to those described earlier in this review.

The studies of DeMott and Davis (47, 48) give a good description of some of the responses seen in chorioretinal burns. Immediately post-trauma (5-30 minutes), visible lesions consisted of intrusions of fluid into the sclera, choroid, and retina. Intrusions were most often observed between the receptor and bipolar cell layers of the retina. From 5 to 36

days post-trauma, the fluid intrusions gradually coagulated and shrank, with resorption of the clot completed by the end of this period. Necrosis of retina appeared in the center of the lesion and there was retinal detachment. In some of the less severe cases, there was a tendency for the retina to re-attach in the healing process. However, in those cases where the intrusion is between the receptor and bipolar layers, the functional loss should be permanent. As intensity of irradiance increased, it was noted that pigment cells and retinal elements were immediately destroyed. At even higher intensities, bipolar and ganglion cell layers were destroyed. Even if not directly involved, these latter cells will show a secondary degeneration if the receptors they synapse with are destroyed. Such retrograde degeneration would in all probability eventually be seen through the entire CNS optical projection involving these receptors and neurons. In summary, DeMott and Davis (48) found that near-threshold lesions could take three forms:

(a) circumscribed retinal detachment, possibly temporary,

(b) separation of bipolar and retinal cells leading to retinal degeneration, or

(c) direct destruction of retinal and neural elements.

They estimated 95% of all threshold lesions would fall in the latter two categories.

3. Case Histories of Damage to the Human Visual System, Nuclear Detonation

There are nine recorded cases of human chorioretinal burns which resulted from viewing a nuclear fireball--directly or indirectly. Occurrences such as these have provided valuable information for correlating human with animal data on threshold levels, symptomatology, and effects on vision. Unfortunately, difficulties in reconstructing the field exposure conditions have restricted the accuracy with which threshold interpretations can be made.

Case 1

The first recorded case was a Japanese survivor of the Hiroshima atomic detonation (31). This person sustained bilateral central lesions. However, irradiance and duration of exposure have not been determined. The greatest value in this particular recorded case (where thousands of survivors were examined) is an indication of the low probability of retinal burn among survivors under the specified conditions at Hiroshima. (That is, constricted day-time pupil, city location where buildings offered many shadows, air burst, low-yield weapon, etc.)

The next body of information on retinal burns was published in 1957 (22). Here, six test personnel were affected who were located at various distances from low altitude or surface detonations. By reviewing the yields for weapons in the 1956 Effects of Nuclear Weapons (78) and noting the distances from detonation (2 to 10 miles) that cases were listed for, it is inferred that the yields were less than 50 KT. It was reported (42) that five of these six cases were not using the recommended eye filters (one of the victims was using a 3% filter). The thermal irradiance values have not been published for these cases so little can be extrapolated about burn thresholds.

Case 2

A photographer viewed the latter part of the fireball through a camera optical viewfinder while flying six miles from the detonation. The left eye was viewing through the camera, the right eye was exposed unshielded. Immediately after detonation, there was a marked dazzle effect followed later by blurring, haziness, photophobia, and "shimmering". These effects persisted sufficiently to prevent the photographer from driving an automobile hours later. Twenty-four hours after exposure, visual acuity was 20/30 O.D. and 20/50 O.S. The eyelids were swollen and conjunctivae were injected. In 48 hours, acuity had increased to 20/15 O.D. Ophthalmologic examination showed the right fundus to be normal, while foveal reflex was minimally

diminished in the left eye. A broad, diffuse relative scotoma was noted in the left nasal paracentral area, but no lesion could be seen.

Case 3

A pilot, located five miles from the detonation, viewed the fireball through an aircraft window, right eye only. The case report states that the pilot was blinded for 15 sec and had only limited vision of instruments for the next 10 sec. It is not stated if this blindness refers only to the affected eye, or was apparent in both eyes despite the fact that the left eye had been covered.

The hazy vision persisted for about 10 minutes. Seven months later, acuity was 20/15 in each eye. A paracentral 2^{o} absolute scotoma was noted in the upper temporal quadrant, surrounded by a 5^{o} relative scotoma, but no lesion was detectable ophthalmoscopically.

Case 4

Another pilot, 10 miles distant from a fireball, commenced observation 50 ms after detonation.* Viewing was with the left eye only, no protective filter. Subjective visual difficulties lasted for about 5 minutes. Five days after exposure, acuity was 20/15 each eye; identical to acuity before the nuclear test. A 2^{o} scotoma was located proximal to the fovea. Interestingly, the lesion occurred on or near a retinal artery, and it was observed that blood flow had been diminished in that artery distal to the lesion. The blanched area was still edematous after 12 days, but one month after exposure the retinal edema had disappeared.

Case 5

An airman, located 10 miles from detonation point, viewed the fireball with both eyes unprotected. He immediately blinked and turned away and did not note any subjective symptoms. Bilateral 5^{o} lesions were discovered two

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*It is not clear how this exact time interval was determined.

months later. The lesions were oval, slightly depigmented along the edges, and located between 5^{o} and 10^{o} from the macula. Visual acuity was 20/25 in each eye. No changes were noted in the succeeding 18 months.

Case 6

An officer, 7 miles from the detonation point, viewed the fireball with one eye. The right eye was covered and the left eye was protected with a 3% transmission filter. Immediately after exposure, the officer noted a central blind spot in his left eye. A 4^{o} paracentral scotoma was mapped. Ophthalmoscopic examination revealed a lesion with elevated yellowish-pink margins and visible gray sclera showing through the center. No hemorrhage was observed, although the area around the lesion was slightly edematous. Three months later the scotoma and lesion remained as before. After one month, acuity was 20/20 corrected, each eye.

Case 7

An officer 2 miles from a detonation point viewed the fireball with his left eye only. Just after exposure, acuity for the exposed eye dropped to 20/200. Six weeks later acuity was 20/70. The scotoma was located centrally and extended 5^{o} by 8^{o}. Ophthalmoscopic examination of the lesion showed tension lines radiating out from the lesion, which could indicate the possibility of future retinal detachment. The lesion was surrounded by an edematous area and was itself of a "brownish color with a pigmented spot in the center".

There are several important aspects of these test personnel case histories. First, only two of the six lesions involved the fovea although all of the individuals "viewed" the fireball. This may indicate a delay factor in foveal fixation. Whether the initial, parafoveal, intense stimulation has an aversive quality which tends to inhibit foveal fixation can only be speculated. Such a reflex effect would only be of significance for detonations which persisted for periods approaching a second or more. Second, these cases

illustrate the necessity for making a distinction between presence of functional impairment and absence or presence of visible lesions. In Case 2, for example, a large scotoma was mapped without ophthalmoscopic evidence of a lesion immediately after exposure. Case 3 showed both relative and absolute scotomas, but no visible lesion when examined 7 months later. Conversely, distinct lesions have been noted close to the macula, but foveal acuity has returned to normal when the edema subsided and the lesions became stabilized. Third, in two of the above cases the lesion occurred on or near a blood vessel and vascular changes were noted in the vicinity of the damage. Pertinent to this observation, [illegible] tion about the rate and extent of repair of a retinal area (secondary damage) as a function of the vascular state. That is, the retinal vessels in patients with diabetes, high blood pressure, atherosclerosis, etc., would not be expected to respond as well to traumatic lesions as would normal vessels.

Cases 8 and 9 occurred during the 1962 Fish Bowl Series (3). The particular detonation which produced the injuries was a very high altitude night shot. The burns were sustained at a slant range of about 30 miles. Neither individual had his protective goggles on during the detonation. The pulse characteristics of this particular detonation were such that the peak irradiance was achieved in a small fraction of a millisecond, and had trailed off to low levels well before a blink reflex could occur. Peak irradiance (3) at the ground station was between 2 and 3 watts/cm^2. This means that the blink reflex would have been of no protective value and that the injured individuals had to be fixating at the exact detonation point when the detonation occurred. One case does give evidence which suggests that the eyeball may have been [illegible] phase of the fireball, since a small tail-like extension was observed on the lesion (3). However, there is also a remote possibility that the two burn victims could have been burned by a specular reflection rather than the direct image. Such reflections could occur from a wristwatch face or any of a variety of shiny metal or glass surfaces.

The clinical data for these latter two burn cases is fairly typical except that the damage to central vision was more pronounced than the six cases cited previously. In the first case, acuity for central vision was 20/400 initially, but returned to 20/25 by six months. The second victim was less fortunate as central vision did not improve beyond 20/60. The lesion diameters were 0.35 and 0.50 mm, respectively. Both individuals noted immediate visual disturbances but neither was incapacitated. In a recent review (42) of these cases, the fact that chorioretinal burns on or near the fovea do not necessarily cause complete blindness was emphasized. Both size and location of the lesion determine visual impairment.

The functional significance of permanent retinal damage is dependent upon two, and possibly three factors: the size of the lesion, the location of the lesion, and subsequent physiological secondary reactions around the lesion site. The first two points are obvious and will be expanded upon below. However, the third factor is one about which little factual information is available. The question here is: will there be delayed or progressive deleterious effects on vision following subsidence of the initial inflammatory reaction after receipt of a chorioretinal burn? In one of the cases of human retinal burns (159), the examining physicians noted the presence of tension lines around the lesion, and suggested that this could indicate the possibility of future retinal detachment. Similarly, Geeraets noted* having seen, in experimental animals, stress or tension lines in the vitreous humor following retinal burns. Whether these isolated examples are indicative of a complication not previously expected will remain to be confirmed by long-term observations.

Returning to the significance of the size and location of retinal lesions, the lesions seen in the accidental human exposures have all been found on or near the fovea. Burns directly on the fovea produce reductions in acuity,

*Personal communication.

the extent depending upon the lesion area. For example, it was calculated, considering only the theoretical geometry of the human eye and measures of relative acuity in peripheral fields of the retina, that a lesion covering 2.5^{o} (1.25^{o} on each side of the fovea) would reduce acuity to 57% of normal, or 20/35. If the lesion covered 20^{o}, acuity would only be 15% of normal, or 20/130 (43a). The fact that the theoretical residual acuity is not always obtained (Case 9, for example) may indicate that the damaged area exceeds the area of the visible lesion, or that some individuals have difficulty learning to "look around" a central scotoma.

Burns located off the fovea will produce blind spots, which subjectively may not be apparent to the individual. However, the visual defect resulting from a parafoveal lesion may not be confined to the lesion area, depending upon the types of cells which are involved. If damage is confined to receptors (mostly rods), the blind spot will be at the lesion site. If nerve fibers are damaged, that portion of the visual field served by those fibers will show a defect. As the axons progress from the periphery of the retina to the optic disc, they form symmetrical bundles. The only exception to the symmetry of these radial fibers and bundles occurs between the macula and the disc. These fiber bundles leaving the fovea are more delicate (154), and therefore, their thermal damage threshold may be lower than the threshold for extrafoveal axon bundles. However, because of the construction and nonpigmented character of nerve fibers, their exposure threshold should be considerably higher than for receptor cells and the pigmented retina. This value is not known.

In the literature reviewed for this report, no description of burns on the optic disc were found. If a burn did occur on the disc, it could cause direct damage to large groups of axons or to retinal blood vessels. Hemorrhage from ruptured vessels could increase ocular pressure, obscure vision by loosing formed elements into the vitreous, and cause neural anoxia distal to the lesion site. There would seem to be a low probability that the

fireball image would fall upon the disc since the individual normally does not focus objects in that location. Also, the absorption of light energy is less for these tissues. It might be worthwhile, however, to examine this question experimentally, if for no other reason than to reassure the investigator that optic disc burns are improbable and/or difficult to inflict.

One of the most significant conclusions derived from observations of the accidental human retinal burn cases is the fact that even with foveal lesions, the individual eventually regains a good portion of his visual acuity (42, 159). Probably the eye scanning patterns are changed slightly to bring objects to focus on undamaged portions of the fovea. On the other hand, the immediate sensations of visual disturbances reported by individuals who have sustained retinal lesions would impede the performance of all but gross visual tasks.

In summary then, the individual sustaining a retinal burn may be effectively visually incapacitated for an hour or more. Barring possible long-term effects, such as retinal detachment or contractions of the vitreous, the visual field defect will eventually subside to an area about the size of the original burn. One of the pressing questions concerns the problem of visible lesion. Are alterations in retinal proteins or enzyme activity responsible, and can this condition be reversed?

D. Mechanisms Involved in the Absorption, Transformation, and Dissipation of Energy

1. Steam Production, Shock Waves, and Mechanical Disruption

Injury mechanisms for suprathreshold doses are believed to differ from those for near threshold doses. If the rate of energy delivery is sufficiently high, such as may readily be obtained with lasers, tissues may be heated sufficiently to vaporize intracellular and extracellular fluids.